THE VETERINARY RECORD

FOUNDED BY WILLIAM HUNTING, F.R.C.V.S., 1888

No. 24. JUNE 12th, 1954 VOL. 66.

Atrophic Rhinitis

Following confirmation of a single case of atrophic rhinitis in a young boar on a farm at Denham, Buckinghamshire, the Ministry of Agriculture and Fisheries has made an Order (The Atrophic Rhinitis Order, 1954) to cover further possible outbreaks. Details of this Order were quoted in full in our issue of June 5th, 1954, p. 329. We trust that the following article will be of topical interest and value to members of the Profession.

The first reported instance of this dreaded disease in Great Britain occurred in circumstances which would appear to justify completely the traditional attitude of veterinary authorities towards importations of livestock. The seven-months-old boar in question was the progeny of one of a consignment of Swedish Landrace gilts, the importation of which was permitted during 1953. It was purchased during February, 1954, when 18 weeks of age and was then found to weigh only 50 lb. A few days after purchase, the new owner noticed that breathing was somewhat laboured and that there was some degree of cyanosis of the ears. This was followed by a type of sneezing suggestive of nasal obstruction, and by a slight haemorrhage from one nostril. Veterinary advice was then sought, and the animal was eventually moved to the Veterinary Laboratory, Weybridge, where a final diagnosis of atrophic rhinitis was made and confirmed in consultation with Swedish veterinarians. Prior to removal, a commencing facial distortion was evident. It is understood that the 1953 importations had all been third litter pigs, the dams in each case having produced two previous healthy litters, and had been subjected to quarantine prior to their despatch from Sweden and again on arrival in this country. The disease is, however, of established occurrence in Sweden, where strict control measures, including slaughter of affected pigs, are enforced. The fact that a case has occurred in the progeny of imported stock, despite strict quarantine measures and every attempt to ensure healthy animals, reflects the insidious nature of the disease and perhaps the wisdom of those who advocate that the upgrading of British stock may be secured more satisfactorily, and certainly with a greater degree of safety, from the progeny testing and selection of our present breeds.

It may be of assistance to practising veterinary surgeons to provide a brief résumé of published information on atrophic rhinitis. Should further cases occur it is of the utmost importance that they should be reported with the minimum of delay. Fortunately the Denham case has attracted a great deal of attention in farming circles, so that veterinary surgeons are likely to be called in immediately a suspected outbreak is observed.

Historical Aspects

According to Thunberg & Carlström (1940), atrophic rhinitis was probably described in Sweden as long ago as 1842. It was certainly known in Sweden later in the 19th century, and has been recognised for many years in Denmark. It was reported in Alberta in 1932, and in Indiana in 1944, and during the past decade it has assumed considerable importance in Western Canada and in parts of the U.S.A. It appears to have been recognised in Germany and some Central European countries since long before the second World War. In the light of the conflicting views on its aetiology, and of the manner in which the first reported British case has developed, it is not surprising that the exact method of spread from one country to another is unrecorded.

Symptoms

The lay observations on the Denham case are mentioned above. A further feature was the continued failure of the boar in question to make normal weight gains, since at 25 weeks of age it scaled 106 lb. Duthie (1947), who had been the first to record the disease in Canada, states that in the early stages the nasal passages are partially occluded due to swelling of the membranes. This may be accompanied by a slight watery discharge from the nose and eyes. There is repeated sneezing, and violent attempts to rub the snout against other animals or objects. If the affected animal has access to soil, it attempts to bury its snout in the earth, presumably to secure relief from pain. Suckling pigs may contract the disease, in which case they tend to make snoring or gurgling sounds as they drink. A tendency shown by many affected animals is that of chewing the tails of other pigs.

The disease does not appear to be fatal during its acute phase, which passes into the chronic. The nasal discharges become progressively more mucoid or mucopurulent, and much scantier in amount. There may in fact, be periods during which very little discharge is observed, but these appear to coincide with accumulations of material in the nasal passages, and at intervals there are paroxysms of violent sneezing during which inspissated matter is expelled. Portions of the turbinate bones, still partly encased in their membranes, may be voided in this manner. In other cases, the discharges form dry adherent crusts or caseous masses within the greatly widened nasal passages. The progressive damage is accompanied by frequent haemorrhage from one or both nostrils, and this is regarded by Duthie as perhaps the most characteristic and diagnostic symptom.

The above processes are accompanied by arrested development of the upper jaw, and ultimately by almost complete destruction of the turbinates. The nasal bones—which, with the premaxilla, show partial decalcification—tend to turn upwards from the lower jaw. Since the growth of the lower jaw is not directly affected, the animal becomes progressively prognathic and dental apposition is poor or lost altogether. On wet feeding systems, bodily growth may proceed even at normal rates, but with dry feeding much difficulty is experienced, the particles of meal may provoke violent sneezing, and actual

starvation may occur in the face of competition from normal pen-mates.

The progressive distortion of the face is characteristic. When the condition is unilateral, a twisted appearance results, whereas in bilateral cases the snout becomes completely "dished." There is a progressive wrinkling of the skin covering the distorted areas.

Pathology

The pathology of Canadian cases has been described not only by Duthie but also by Schofield & Jones (1950), whose paper should be consulted for details of the gross pathology and of the histological changes observed in the mucous membranes, blood vessels, periosteum and bone.

Suspected Inherited Susceptibility

An inherited susceptibility to atrophic rhinitis has long been suspected, and one German worker postulated in 1878 that a congenital under-development of the nasal muscles was a contributory factor. The evidence up to 1949 has been examined by Gilman, who feels that extreme caution should be exercised in associating any inherent differences in conformation with the incidence of rhinitis. Gilman himself, however, found that whereas there was no evidence of a relationship between short nose and susceptibility within the Yorkshire breed, there was a significant difference ($P = 0{\cdot}02$) between the mean nose length of susceptible and resistant Tamworth pigs. Until other aetiological factors have been evaluated it will be difficult to assess further possible hereditary or congenital influences.

Attempts at Transmission

Various attempts to reproduce the disease by the inoculation or instillation of suspected infective material have given somewhat conflicting results. Phillips (1946) recorded the reproduction of an atypical rhinitis in baby pigs through the injection of a bacteria-free filtrate into the nasal cavities, and considered that secondary organisms played an important role in the destructive processes seen in natural cases of the disease. Other earlier attempts to transmit the disease are reviewed by Gwatkin, Plummer, Byrne & Walker (1949), who themselves recorded that the instillation of washings from adult pigs affected with rhinitis resulted in the partial or complete disappearance of turbinated structures in baby pigs, the condition being fairly definite at 30 days, and that from these baby pigs in turn the infection could be passaged. No definite symptoms were observed, however, and the condition of the turbinates was confirmed only on *post-mortem* examination. Spindler, Shorb & Hill investigated the possible role of trichomonads, finding that an atrophy of the turbinates developed in piglets inoculated with nasal or caecal washings containing these protozoa, irrespective of whether they had been obtained from normal or rhinitic animals. Carter & McKay (1953) found that some of the bacteria hitherto associated with atrophic rhinitis were pleuropneumonia-like organisms, and believed that they might be of aetiological significance, particularly in combination with *Fusiformis necrophorus*. Switzer (1953) claimed to have isolated a filterable agent from a suspension of mucous membrane of affected animals, and to have infected young pigs and also chick embryos. He has not so far recorded, however, the successful passage of chick embryo material into pigs. Gwatkin, Dzenis & Byrne (1953) stated that they had reproduced rhinitis in baby pigs by the nasal instillation of pure cultures of *Pasteurella multocida*, Type B, isolated from a field case, and that there was some indication that a filtrate of nasal material increased the activity of the organism.

It seems clear that much further work remains to be done before the role of possible infectious agents can be established, particularly since the experimental conditions that have been reproduced are not clinically identical with field cases. In view of the fact that the disease is now notifiable in Great Britain, the somewhat limited published observations on therapy are not reviewed.

References

Carter, G. R., & McKay, K. A. (1953). *Canad. J. comp. Path.* **17.** 413.

Duthie, R. C. (1947). *Canad. J. comp. Med.*, **11.** 250.

Gilman, J. W. P. (1949). *Ibid.*, **13.** 266.

Gwatkin, R., Dzenis, L., & Byrne, J. L. (1953). *Ibid.*, **17.** 215.

———, Plummer, P. J. G., Byrne, J. L., & Walker, R. V. L. (1949). *Canad. J. comp. Path.*, **13.** 15.

Hoflund, S. (1937). *Svensk. svin. Tidskr.* **9.** 206.

Phillips, C. E. (1946). *Canad. J. comp. Med.*, **10.** 33.

Scholfield, F. W., & Jones, T. L. (1950). *J. Amer. vet. med. Ass.*, **116.** 120.

Spindler, L. A., Shorb, D. A., & Hill, A. B. (1953). *Ibid.*, **122.** 151.

Switzer, W. P. (1953). *Ibid.*, **123.** 45.

Thunberg, E., & Carlström, B. (1940). *Skand. vet-Tidskr.* **48.** 711.

. . . . . .

New Ministerial Orders

As a further precaution against the spread of atrophic rhinitis in this country, the Minister of Agriculture and Fisheries has made Orders prohibiting the import of pigs, except for immediate slaughter, from the Channel Islands and Northern Ireland. Both the Channel Islands and Northern Ireland have recently imported Landrace pigs from Sweden. Some progeny of Landrace pigs which were imported last year into Great Britain are also now in Northern Ireland.

The Orders came into operation on June 2nd.

HUMANE RABBIT TRAP—RESULTS OF TRIALS

The Minister of Agriculture and Fisheries in a Parliamentary answer on June 3rd gave details of the results of a large-scale test by County Agricultural Executive Committees in England and Wales of the "Imbra" humane rabbit trap during the winter and spring, and trials by the Department of Agriculture for Scotland, compared with gin traps. A fairly high proportion of the "Imbra" traps caught rabbits on most locations after experience in handling the traps had been gained, and the rabbits were nearly always killed outright. The "Imbra" appears to give its best results in unobstructed burrows and on dry soil. In the comparative trials in Scotland, fewer rabbits were caught with the "Imbra" trap in its present form than were caught in gin traps.

The Pests Bill, which is now before Parliament, makes provision for abolishing the gin trap and for the approval of humane traps for use in its place. As the Minister has announced, it is proposed to fix July 31st, 1958, as the date after which the use of gin traps shall be banned, and he and the Secretary of State are setting up a Humane Traps Advisory Committee to speed up the development of humane traps and to advise on their approval. One of the first tasks of the Committee will be to consider whether the "Imbra" trap should be recommended for approval when the Bill becomes law.

A MODERN APPROACH TO CANINE VIRUS DISTEMPER AND ITS PRACTICAL APPLICATION*

BY

N. M. LARIN

AND

S. F. J. HODGMAN

Canine Research Station of the Animal Health Trust, Kennett, Nr. Newmarket, Suffolk

The Disease

Definition

On reading the definition of canine distemper (Carré, 1905a and b, or Laidlaw & Dunkin, 1926b), one is forced to conclude that the typical "old fashioned" distemper has changed. According to Geiger (1939) "canine distemper is an acute communicable disease of the dog and certain other carnivorous animals, which is caused by a virus in co-operation with certain bacteria and which is characterised by catarrhal inflammation of the orbital mucous membranes, respiratory and digestive organs, and which is sometimes followed by severe nervous manifestations." Accepting this as the typical form it is obvious that atypical forms are now more generally seen.

It is common nowadays to use the name "hard pad" disease or "para-distemper" in suspected cases of distemper which do not manifest the "classical" signs. Some authors, however, consider the name "hard pad" disease a misnomer, because hardening of the pads is neither a new nor constant sign—having been observed in varying degrees in so-called "classical distemper." On the other hand, the hyperkeratosis is sometimes hardly recognisable even histologically. The name "para-distemper" is, in the opinion of the authors, still more unsatisfactory because this disease has never been defined.

MacIntyre, Trevan & Montgomerie (1948) reported that encephalitis is the most important syndrome of "hard pad" disease. Others have also written on this subject (Singleton & Potter, 1949; Wilkinson, 1950; Scheitlin, Sieferle & Stunzi, 1951). The high incidence of canine distemper virus in cases of canine encephalitis (82·1 per cent.) has recently been reported by Ribelin in the U.S.A. (1953).

Epizootology

As in other infections the epizootology of canine distemper is concerned with three factors, host, infectious agent, and the mechanism of getting the two together. Once an outbreak of the disease has started it is simple to explain its continuation on the basis of spread either by direct or indirect contact. The difficulty is to establish where and how the initial case started.

In canine virus hepatitis recovery from the clinical illness may not be accompanied by the disappearance of the causative agent, and inapparent infections are also very common. Thus, it can be easily visualised how infection may arise from exposure to sub-clinical cases or to virus carriers (Larin, 1953). Another type of epizootological set-up in which a silent infection in one species spreads to another as a highly fatal disease, is that exemplified by bovine pseudorabies.

In swine influenza virus we have a cold-blooded intermediate host, the swine lung worm, which adequately accounts for the survival of the virus. The carriers or intermediate hosts in distemper remain unrecognised.

It seems that distemper epizootics exhibiting a high fever with involvement of the respiratory and digestive organs run a fairly rapid course and do not leave carriers. On the other hand, the disease where involvement of the nervous system is the most prominent syndrome, frequently lingers for months. In four cases of clinically "recovered" canine encephalitis we isolated distemper virus from the brain and spinal cord, but did not find it in the blood and other organs. Whether it may be in such cases that the virus can be shed under the stress of another infection, etc., remains to be answered.

The high incidence of active immunity among adult dogs in which signs of the disease have not been observed, suggests that they acquired the immune state as the result of clinically inapparent infections and hence they could be a source of infection.

Factors complicating virus distemper

According to Whitney & Whitney (1953) the disease cannot be fully comprehended if one considers the canine distemper virus alone. Although the dog is probably more generally resistant to infections than are human beings, the hygienic conditions under which it lives differ from those of man. There is ample evidence that the dog can suffer simultaneously from more than one virus infection, not allowing for rickettsia, bacteria, protozoa, and parasitic worms, any of which can invade the body either prior to, simultaneously, or after virus infection.

The presence of canine hepatitis virus, or the damage produced by it, can facilitate and aggravate a simultaneous or subsequent infection with distemper virus, and considerably change the clinical picture of distemper (Gillespie, Robinson & Baker, 1952; Larin, 1953). Distemper can also occur either simultaneously with leptospirosis, or one can be superimposed on the other. Until a few years ago only two canine virus diseases were recognised, rabies and distemper. To-day, it seems to be more than a probability that the dog may represent a larger reservoir of virus infections than was at one time thought.

The same is true for bacteria. There are about 20 species known which can complicate virus distemper. Although these are often called "the secondary invaders" some of them, *e.g.*, streptococci, may inhabit the body prior to distemper virus infection.

Pathology

Pathological findings in canine distemper vary considerably subject to the variations in the disease and complications occurring. This is the reason for there being no specific lesions.

The virus has an affinity to epithelial tissue, so that membranes of the vascular, respiratory, digestive and genito-urinary organs are usually the first affected. In a dog killed in the early stages of the disease there is little, if anything, to be seen macroscopically; the most prominent microscopical lesions are swelling of the mucous membranes and some areas of cellular necrosis.

In the later stage, gross pathology often shows inflammation of the gums, pharynx, larynx and tonsils. Pleura and lungs may show various degrees of inflammation. The spleen is seldom much enlarged. There can be marked swelling and congestion of mesenteric lymph nodes. The gastro-intestinal tract is inflamed and sometimes ulcerated. The liver and renal-cortex may show degeneration. In some cases the respiratory organs are more severely damaged than the gastro-intestinal tract, etc., and vice versa. These variations often depend on complicating factors and the treatment used.

* Abridged version of a paper given to the Midland Counties Veterinary Association on February 24th, at Birmingham.

Nervous damage according to MacIntyre *et al* (1948) differs in distemper to that seen in "hard pad" disease. The damage in distemper is degenerative and not neurotropic with no demyelination. In "hard pad" the brain lesion is essentially a demyelinating encephalitis occurring in small scattered areas, typically in the cerebellar peduncles and folia.

Cerebro-spinal fluid in distemper can change from water-clear to pathological, and globulin reaction is strongly positive (Luque, 1950).

The Virus

History

Carré's discovery that canine distemper was caused by a virus was finally accepted through the work of Laidlaw & Dunkin (1926c), who proved that distemper was a virus disease and introduced the use of ferrets as experimental animals.

It was thought that distemper virus represented one etiological entity and an attempt was made (Holmes, 1948) to arrange it with other animal viruses into species, genera and families, and to give them internationally recognised names. In consequence, the virus received the name *Tarpeia canis*, and undue emphasis was laid upon some of its general characteristics which implied the extreme simplicity of the virus structure and a lack of any means for its change and evolution. Over the years, however, research has revealed the complexity and diversity of its character, and has attracted an active interest by both the veterinary and medical professions.

Susceptible species.—A new and interesting aspect of the virus is the expansion of susceptible species. Benner (1931) reported that after the inoculation of distemper virus to pigs, they developed signs of swine influenza. Harispe & Wainey (1942) isolated the virus from pumas, and Piat (1950) from young lions, thus establishing the susceptibility of the big cats. According to Goss (1948) apart from the dog the following mammals are susceptible to the virus : buiturong, dingo, fox, coyote, wolf, racoon, weasel, ferret and mink. Martin (1950) described the transmission of ferret adapted distemper virus to monkeys. Transmission of the virus to rabbits was reported by Goret, Martin & Jaubert (1950). Martin (1953) also reported the transmission of the virus to gerbils and white mice.

Transmission.—By contact, and probably by airborne droplets. No arthropod vector is recognised (Holmes, 1948).

Size of virus particle

The virus passes Chamberlain L_2 and Mandler's filters. Electron-micrographs of concentrate of a commercial vacuum-dried active virus show virus-like particles which are uniform in size and may be canine distemper virus. These particles are round and have a mean diameter of 21 millimicrons by direct measurement of electron-micrographs (Reagan & Brueckner, 1951). Thus it would seem that in a range of animal viruses particulate in size varying from 250 to 10 millimicrons, canine distemper belongs to the smallest viruses.

Stability

According to the literature reviewed by Holmes (1948) the virus survives :—

In wet tissue at 10° C. for 35 not 85 days ;

In glycerine-saline solution at 10° C. for 67 days ;

In 25 per cent. horse serum at −24° C. for more than 693 days ;

In vacuum desiccated tissue at 10° C. for 90 days ;

In freeze dried tissue, (*a*) sealed in oxygen-free nitrogen for 365 days at 70° C., (*b*) sealed in vacuum at least 430 days at 7° C.

On the other hand, the virus can perish within a few hours on being exposed to light at room temperature or to the usual disinfectants.

The virus has been recovered from carcases of dogs 48 hours after death with signs of slight *post-mortem* deterioration, but attempts made to recover the virus from carcases with advanced decomposition were unsuccessful (Larin, 1945b). The latter can probably be explained by experiments conducted by Bindrich (1951) who established that the optimal *p*H value for distemper virus is 7·0. The virus is fairly resistant against more acid *p*H values but is quickly destroyed if *p*H is on the alkaline side. Therefore, 3 per cent. sodium hydroxide appears to be a rapid and effective disinfectant for the virus.

Antigenic properties.—Some cross immunity between canine distemper and human influenza viruses was reported by Eichhorn & Pyle (1934), and a certain immunological relationship between canine distemper and poliomyelitis viruses by Dalldorf, Douglass & Robinson (1938) ; Anon (1948), and Martin (1949). Reagan & Bruechner (1952) described a certain resistance produced by the Newcastle disease virus to distemper virus.

Propagation in embryonated hen eggs.—Several attempts have been made to adapt the virus to tissues of developing chick embryos, but some of them have not been particularly successful (Mitcherlich, 1938 ; Plummer, 1939 ; Beveridge & Burnet, 1946 ; Haig, 1948). Finally, the virus was attenuated and maintained through 74 passages on the chorio-allantoic membranes of developing chick embryos (Cabasso & Cox, 1949).

Propagation in tissue cultures.—The virus was propagated in cultures of young dog tissues such as spleen, lung, lymph nodes and testes (Dedie & Klapötke, 1951). Using chick embryo tissues, the virus isolated from a ferret was cultivated *in vitro* over a period of six weeks (Lasfargues & Corvazier, 1952).

Inclusion bodies.—There is evidence that the inclusion bodies can be present in the cytoplasm and the nuclei of the cells. Lasfargues & Lasfargues (1952) demonstrated strongly eosinophilic inclusion bodies in chick embryos tissue cultures inoculated with the virus. The inclusions were not, however, regularly produced in subsequent passages.

Serological reactions.—A complement fixation test for the evaluation of hyperimmune serum was reported by Laidlaw & Dunkin (1931) and afterwards by Pyle & Brown (1935), Goret, Bardach & Lélandais (1936), and Mitcherlich (1938). Dedie (1951) and Dräger & Schindler (1951) described a complement fixation test for the diagnosis of distemper. Mansi (1952) used a complement fixation test for serological studies of different strains of distemper virus.

Haemagglutination reaction with distemper virus using human or frog erythrocytes was described by Vladimirov (1949). Corvazier & Lasfargues (1952) described the conglutination test which was used to determine the presence of either the virus in tissues or the antibody in immune serum.

Purification of virus.—Larin (1954a) described two methods for purification of the virus from large amounts of host protein in tissue suspensions.

The complexity of the virus

The first intimation that strains of distemper virus might have important antigenic differences came when Laidlaw & Dunkin (1928a) reported their studies on the immunisation of ferrets. Verlinde (1946) demonstrated in a form of canine

distemper a virus which immunologically differed from the known Carré virus. MacIntyre *et al.* (1948) stated that recovery after either Laidlaw-Dunkin or " hard pad " virus infection, left the animal immune to the homologous virus only.

Koprowski *et al.* (1950) stated that canine distemper virus was responsible for epizootics of demyelinating encephalitis in dogs.

Gillespie (1951) found that of three strains of distemper virus studied, catarrhal without encephalitis, " hard pad " and encephalitis, and a commercial strain from Fromm laboratories, were indistinguishable in the cross immunity tests.

Mansi (1952) conducted a comparative study using a strain of Laidlaw-Dunkin virus and a strain of " hard pad " virus obtained from MacIntyre and co-authors (1948), also a strain of distemperoid virus and the neurotropic strain of distemper virus Glasgow S123 (Mansi, 1951). All these four strains were found to be indistinguishable in the cross-immunity tests and by his method of complement fixation reaction, although the course of infection differed in the ferret.

It is noteworthy, however, that the neurotropic strain Glasgow S123 has changed after as few as five consecutive passages and produced the syndrome similar to that obtained after inoculations with Laidlaw-Dunkin virus (Mansi, 1952).

When we commenced our studies on the virus in 1952 we felt that our first step should be the isolation and identification of current strains of the virus from cases of clinical distemper in various parts of Britain. In attempting to isolate as many strains as possible, we used adult healthy ferrets and tried to introduce uniformity in all our procedures.

A preliminary report on the study of 23 strains of virus has been published (Larin, 1954a). It was found that the behaviour in the ferret of these strains was so strikingly different as to justify their groupings as follows :—

Group	A	B	C
Number of strains	4	6	13
Incubation period	3–6 days	3–10 days	3–10 days
Duration of illness	6–14 ,,	2–6 weeks	3–12 weeks
Febrile reaction	Either continuous or diphasic fever often with hyperpyrexia (106° F.)	Relapsing fever	Relapsing fever with moderate elevations of temperature and sub-normal temperatures during remissions.
Mouth lesions, conjunctivitis, diarrhoea	In the majority of ferrets	Inconstant	Inconstant
Anorexia	Only during one or two days before collapse and death	Frequently occurs during the illness	Very frequent during the illness, probably stimulates the fatal outcome
Hardening of the pads	0	0	0
Nervous manifestations	0	0	0
Mortality	Fatal for all inoculated ferrets	About 70 per cent.	About 50 per cent.

A group strains have not changed during up to 20 consecutive passages in the ferret. B group strains produced variants belonging either to A or C group. C group strains produced B variants but, so far, no A variants. It would also appear from the above table that the ferret is not a highly susceptible host for successfully propagating C group strains.

Some ferrets which recovered after inoculation with either C or B group strains had no immunity to A group and died after being challenged, while others developed febrile reactions but recovered. Simultaneous inoculations of A and C strains to ferrets produced the disease characteristic of A group only. Infection with A, B and C group strains can occur at the same time in a single epizootic.

We would like to emphasise the fact that we are not trying to associate any particular type of virus with a particular form of clinical distemper. Neither do we propose our classification for general use. Our research on this subject is still in progress, and the above data must be regarded as an attempt to build up a tentative and very incomplete picture of distemper virus-complex.

It can be concluded, however, that progressive and continuous changes have been occurring in strains of distemper virus. The differences in the strains are sufficiently great to assume the plurality of the virus and to warrant periodic revision of the components in immunising materials in order that the strains used may closely resemble the current strains infecting the dog. It should also be realised that canine distemper is not caused by a homogenous virus possessing distinctive characteristics, but by several types, which are, moreover, far from being stable entities with predictable behaviour. They change and may produce new forms of a modified nature and of a different biological behaviour.

Diagnosis

The specific diagnosis of the disease is far from being simple. Serological tests are still in a stage of development. The study of the virus in tissue cultures is only in its embryonic stage. Propagation of the virus in embryonated avian eggs is a too lengthy procedure when considered as a method for diagnosis. As there are no pathognomonic lesions in this disease, a pathological diagnosis is greatly fortified when characteristic inclusion bodies are found. Thus, the diagnosis of the disease must be based largely on clinical manifestations, pertinent history, and on the use of ferrets when a more conclusive identification is required. The virus, however, is not one of those that constantly invades the blood stream. Neither is it constantly present in the discharges. This has to be borne in mind when a laboratory diagnosis is to be made on a living dog with clinically suspected distemper. The plurality of the virus also demands persistent consideration in the approach to the diagnostic problem.

Immunity

A discussion on immunity to distemper virus may be approached from several angles, but will probably raise more questions than can be answered with finality.

The mechanism of immunity

Although research over the last few years has elucidated the mechanism of immunity response in virus diseases, we have so far no direct knowledge concerning distemper, and one can do no more than speculate on immunity in distemper. This might be interesting but would also be embarrassing.

There are virus diseases such as poxes, rinderpest, equine encephalomyelitis, and others, in which the virus is essentially a homogenous entity and in which a generalised infection with viremia is an essential part of the disease. A staunch immunity of prolonged duration follows these infections, and the results of research present a comprehensive explanation of the immunity nature from the point of view of the functioning of specific antibodies.

In another group of virus diseases, including human influenza, the common cold, etc., it is commonly agreed that a permanent immunity does not exist at all. There is also no satisfactory evidence in such diseases that the invasion of the blood stream is an essential part of the disease ; the damage to the invaded cells probably accounts for many of the symptoms, absorption of products of damaged cells may account for others. These viruses seldom appear in the blood stream, and so probably escape the challenge of antibodies and leukocytes, and this may be the explanation of repeated attacks.

Another factor seriously influencing the duration of immunity to the same disease is the homogenity of the virus involved. The immunological differences of viruses of human influenza, foot-and-mouth disease, and others, have clearly furnished the explanation for repeated attacks of the same disease.

But what of canine distemper ? Some forms of the disease do produce a generalised infection with viremia, and it is said that animals which recover from such an attack are solidly immune for life. This suggestion, however, is not sufficiently supported by statistical evidence. On the other hand, there is evidence that invasion of the blood stream by the virus is not an essential part of the disease in some forms of distemper. There is also an increased awareness that immunological types of the virus may obscure the immunity.

Artificial Immunisation

Active immunisation. — There are different practical approaches in producing active immunity to virus diseases. The use of active virus plus antibody involves a certain risk that the virus-antibody equilibrium may not be sufficient to prevent the development of the disease with a possible fatal outcome in animals with a low resistance. This risk, however, is sometimes taken in veterinary practice. In human medicine, where no risk must be taken, the usual approach for active immunisation is by the use of inactivated or attenuated non-virulent viruses.

There is ample evidence that many viruses, when inactivated with low concentrations of formalin or by ultra-violet light, are capable of evoking immunity (Bedson, 1937 ; Olitsky & Casals, 1945). But it is true that the immunity evoked by inactive virus may not always remain as long, or be as solid, as that produced by an active virus. Therefore, vaccines made from inactivated virus are sometimes used to establish sufficient immunity to permit a later injection of active virus to make the immunity more solid. Laidlaw & Dunkin (1928b) made use of such a procedure in immunising dogs against distemper.

When a dog is to be actively immunised against distemper the veterinary surgeon concerned must consider which approach to choose. In the case of sporting dogs, Army dogs, etc., the first approach using virus-serum or the vaccine-virus method would probably be acceptable, because in this case the cost of the immunisation, and the high grade of the immunity, is often the principal *sine qua non*. On the other hand, there is the case of the pet dog in which the sentiments of the owner must be considered, the dog perhaps being regarded as one of the family. In such cases, the owner may not wish to take the risk of active immunisation.

There are four methods of active immunisation against distemper used at present : (1) active virus-serum, (2) vaccine-active virus, (3) ferret attenuated virus (distemperoid virus), and (4) avianised virus (egg-adapted virus).

There is, unfortunately, no statistical evidence available to compare which method is the most effective, and we have no experimental data on this subject. It is claimed that distemperoid virus produces a long immunity after a mild reaction which may pass unnoticed. It seems that avianised virus produces no reaction at all, and it is claimed that when such immunised dogs are exposed to the infection they remain resistant.

Avianised virus is a recent development in the active immunisation against distemper, but unfortunately there is very little experimental evidence available on this subject. Cabasso, Stebbins & Cox (1953), in an attempt to determine the minimum interval between injection of the egg-adapted virus and the development of a useful degree of immunity, found that the period was as short as one day ; but the number of experimental ferrets used was small. In still another experiment with a small number of ferrets, the duration of immunity after injection was two years. In the hands of other investigators avianised distemper virus conferred no immunity on ferrets in a small experiment (Lucam, Goret, Jaubert & Poussot, 1953).

Passive immunity

The passive immunity produced by distemper antiserum cannot be expected to remain complete for more than two or three weeks. The recent experimental work of one of the authors (Larin) has shown that the virus antibody is associated with gamma globulin fraction of canine serum. This made possible the purification and concentration of the antibody, which is now being used for further studies. The results of the experimental data on this subject will be published in due course.

Treatment

Nothing very dramatic can be expected from the therapeutic use of antiserum in distemper. By the time the disease is manifested many cells have already been infected, and the virus in their interior is protected against antibody ; so that all one can do by means of serum treatment is to try and prevent, to some extent, the infection of additional cells, and thus hope to minimise the course of the disease.

Chemotherapy

Probably more preparations have been used in attempts to cure distemper than in any other virus disease. But the difficulty of chemotherapy in distemper, as in other virus diseases, lies in the necessity for the inhibiting or inactivation of the virus within the cells without damaging the latter.

It is now clear that the empirical approach will not solve the problem of chemotherapeutical treatment of distemper, and the most promising line is the patient study of the growth requirements of the virus. Armed with this knowledge, substances might be found which were capable of altering the relationship between the virus and the cell in such a way as to allow the cell to survive the competition of the virus.

Summary

In order to understand the clinical picture of naturally occurring distemper, one must realise that a dog can suffer simultaneously from more than one virus infection, not allowing for rickettsia, bacteria, protozoa and parasitic worms. Canine distemper is not caused by a homogeneous virus possessing distinctive characteristics, but by several types which are, moreover, far from being stable entities with predictable behaviour. The plurality of the distemper virus itself may also cause variations in the disease.

The high incidence of naturally acquired active immunity in adult dogs in which signs of the disease have not been

(*Continued on page* 343)

observed, suggests that the infection can run a clinically inapparent course in some dogs, which can thus be a source of infection.

The diagnosis of the disease must be based largely on clinical manifestations, and a pertinent history, with the use of ferrets when a more conclusive identification of the disease is required. The virus does not constantly invade the blood stream, neither is it constantly present in the discharges. This should be remembered when a laboratory diagnosis is required on a living dog with clinically suspected distemper. There is no direct knowledge concerning the mechanism of immunity in distemper, nor is there an easy answer to the problem of active immunisation against the disease. The veterinary surgeon must judge each case according to its merits before making any decision as to the method he intends to adopt and the preparations he will use.

Acknowledgment.—We wish to thank Mr. J. K. Bateman for his valuable co-operation, and also all those veterinary surgeons who have given us the opportunities to investigate outbreaks. Our further thanks are due to our laboratory and animal staff, particularly to Messrs. W. G. Orbell and M. J. Taylor.

References

Anon. (1948). *Rapp. Inst. Maroc.* 21.
Aubert, A. (1950). *Rev. Path. comp.* **50.** 520.
Bedson, S. P. (1937). *Proc. Roy. Soc. Med.* **31.** 1.
Benner, J. W. (1931). *Cornell Vet.* **21.** 1.
Beveridge, W. J. B., & Burnet, F. M. (1946). *Med. Res. Council Sp. Report Series.* **256.** 76.
Bindrich, H. (1950). *Exp. Vet. Med.* **2.** 73.
———. (1951). *Ibid.* **4.** 119.
Cabasso, V. J., & Cox, H. R. (1949). *Proc. Soc. exp. Biol. and Med.* (N.Y.). **71.** 246.
———, Stebbins, M. R., & Cox, H. R. (1953). *Vet. Med.* **48.** 147.
Carre, H. (1905a). *C.R. Acad. Sci.* **140.** 669, 689, 1489.
———. (1905b). *Rev. Gen. Med. Vet.* **5.** 321.
Corvazier, R., & Lasfargues, E. (1952). *C.R. Soc. Biol. Paris,* **146.** 676.
Dalldorf, G., Douglass, M., & Robinson, H. E. (1938). *J. exp. Med.* **67.** 33.
Dedie, K. (1951). *Exp. Vet. Med.* **4.** 127.
——— & Klapōtke, E. (1951). *Ibid.* **4.** 137.
Dräger, K., & Schindler, R. (1951). *Mh. prakt. Tierheilk.* **3.** 262.
Eichhorn, A., & Pyle, N. J. (1934). *J. Amer. Med. Assoc.* **102.** 2082.
Geiger, W. (1939). *Handbuch der Viruskrankheiten.* Haagen, E., and Waldmann, D. **2.** 363.
Gillespie, J. H. (1951). *No. Am. Vet.* **32.** 837.
———, Robinson, J. I., & Baker, J. (1952). *Proc. Soc. exp. Biol. & Med.* (*N.Y.*). **2.** 81, 461.
Goret, P., Bardach, M., & Lelandais, E. (1936). *Bull. Acad. vet. Fr.* **89.** 315.
———, Martin, L. A., & Jaubert, L. (1950). *Ibid.* **23.** 295.
Goss, L. W. (1948). *Am. J. vet. Res.* **9.** 65.
Haig, D. A. (1948). *J. South Afr. Vet. Med. Assn.* **19.** 73.
Harispe, C. M., & Wainey, Y. I. (1941). *Rev. Med. Vet.* (*Buenos Aires*). **23.** 492.
Holmes, F. O. (1948). "Bergey's Manual of Determinative Bacteriology." Supp. 2. 1272.
Koprowski, H., Jervis, G. A., James, T. R., Burkhart, R. L., & Poppensiek, G. C. (1950). *Amer. J. Hyg.* **51.** 63.
Laidlaw, P. P., & Dunkin, G. W. (1926a). *J. Comp. Path. & Therap.* **39.** 201.
———. (1926b). *Ibid.* **39.** 213.
———. (1926c). *Ibid.* **39.** 222.
———. (1928a). *Ibid.* **41.** 1.
———. (1928b). *Ibid.* **41.** 209.
———. (1931). *Ibid.* **44.** 1.
Larin, N. M. (1953). *Trans. 4th Meeting Internat. Soc. Geograph. Pathology Schweiz. Z. Allgem. Path. & Bact., Separatum,* **16.** 3, 614.
———. (1954a). *Nature.* **173.** 174.
———. (1954b). Unpublished data.
Lasfargues, E., & Corvazier, R. (1952). *C.R. Soc. biol. Paris.* **146.** 1713.
——— & Lasfargues, J. (1952). *Ibid.* **146.** 1711.
Lucam, F., Goret, P., Jaubert, L., & Poussot, A. (1953). *Bull. Acad. vet. Fr.* **26.** 67.
Luque, J. M. S. (1950). *Ann. Fac. Vet., Madrid.* **2.** 112. (English Summary.)
MacIntyre, A. B., Trevan, D. J., & Montgomerie, R. F. (1948). *Vet. Rec.* **60.** 635.
Mansi, W. (1951). *Brit. Vet. J.* **5.** 214.
———. (1952). *State Vet. J.* **7.** 22.
Mariotti, P. (1951). *Ann. Fac. Med. Vet. Pisa.* **4.** 69. (English Summary.)
Martin, L. A. (1949). *Maroc. Med.* **28.** 488.
———. (1950). *Bull. Acad. vet. Fr.* **23.** 457.
———. (1953). *Ann. Inst. Pasteur.* **84.** 478.
Mitcherlich, E. (1938). *Dtsch., tierarztl. Wschr.* **48.** 497.
Olitsky, P. K., & Casals, J. (1945). *Bull. New York Acad. Med.* **21.** 356.
Piat, B. L. (1950). *Bull. Serv. Elev. Industr. Anim. A.O.F.* 3.
Plummer, P. J. G. (1939). *Canad. J. Comp. Med.* **3.** 96.
Pyle, N. J., & Brown, R. M. (1935). *J.A.V.M.A.* **87.** 522.
Reagan, R. L., & Brueckner, A. L. (1951). *Cornell Vet.* **2.** 141.
———. (1952). *Vet. Med.* **47.** 513.
Ribelin, W. E. (1953). *Amer. J. Vet. Res.* **14.** 96.
Scheitlin, M., Seiferle, E., & Stunzi, H. (1951). *Schweiz. arch. Tierheilk.* **93.** 91.
Singleton, W. B., & Potter, R. (1949). *Vet. Rec.* **61.** 826.
Teller, H. (1952). *Mh. Vet. Med.* **7.** 339.
Verlinde, J. D. (1946). *Tijdschr. Diergeneesk.* **71.** 210. (English Summary.)
Vladimirov, V. V. (1949). *Veterinariya Moscow.* **26.** 7, 59.
Whitney, L. F., & Whitney, G. D. (1953). "The Distemper Complex." Pract. Sci. Pub. Co., Orange, Connecticut.
Wilkinson, G. T. (1950). *Vet. Rec.* **62.** 453.

General Discussion

In opening the discussion which followed the paper, Mr. J. K. Bateman said : It gives me very great pleasure to open the discussion on this paper so ably presented by Messrs. Larin & Hodgman, and I regard it as a great honour to have been invited to do so. The picture of canine virus distemper is (as you will all have gathered), still a very complicated one and the answers to the complexities may still be a very long way off. So it can do nothing but good when a Station instituted purely for research in canine ailments sets itself the task of endeavouring to eliminate the difficulties inherent to our biggest canine disease.

We have listened to a very comprehensive survey. We have heard of the results and conclusions drawn from their work by various authors. But it says something for the authors of this paper that they take no sides at this stage ; they regard their work as more or less exploratory, and leave us to hope that their future studies may lead to findings of great importance.

My recent personal experiences in distemper are limited to the disease as it affects the greyhound in large kennels, and although we have been told to-day that the virus is always changing I would like to emphasise that, from my experience, the overall picture has changed but little in the last 25 years. I am willing to subscribe to the theory that epizootics may differ from one year to another, or from one epizootic to another in the same year. In one epizootic the disease may take the form of being pronouncedly diphasic with pneumonia and copious nasal discharge as the prominent features ; death rate being comparatively low and nervous disturbance small. In another epizootic, tendency to pronounced secondary rise of temperature is replaced by a long, usually lower, febrile state, or by irregular high febrile reactions accompanied usually by nasal discharge and a higher degree of nervous complications and consequent higher death rate. At any time, however, the type of illness during the epizootic may revert to what the authors to-day call the old-fashioned Laidlaw-Dunkin or Carré disease.

My view—and it is pure speculation—is that the virus is subject to various mutations, that it may change both *in vitro* and in the animal body, and that it is extremely likely that

we have more than one type or mutation present in some sick animals at one time. It would be extremely interesting if it could be ascertained what factors determine the onset of nervous symptoms, and I look forward to the day when the central nervous system can be protected from damage in the distemper-infected dog.

In my experience, recovery from any one of the types of the disease will give immunity to all, and an immunity which is lifelong if the dog continues to lead a normal dog's life, mixing with its fellows some of whom, being infected, will enhance its immunity.

I must say I am intrigued by the authors' query as to where the virus hides between outbreaks. I wonder—does it hide at all? As one who witnesses outbreaks in our own premises every year I know that these start from the entry of an infective dog which has come from infected premises. For some time previous to this entry we may have been free—even up to six months—but during this period we have been accumulating quite a formidable number—perhaps 50—of distemper susceptible young dogs. Once infection is introduced it takes about four to five weeks for all 50 to become smitten. That is how my epizootics occur. In a larger sense and much wider area, field epizootics occur, in my opinion, just that way. The country is never clear. Some dog somewhere has distemper. It may be a pet one which gets loose, or even a fox. It may be spread by indirect contact on clothes of an attendant or visitor, or even by infected ground. At any rate, in its infective state an animal manages to infect, directly or indirectly, another single dog, until one day a dog capable of infecting many young animals takes the disease and an epizootic has begun. Many succumb, but the disease does linger on by passage through individuals who, in turn, manage to spread it to a new crop of youngsters which have begun to grow up.

I do not regard it as terribly mysterious. In support of this view, I maintain that it is not very difficult to keep a place free of distemper for many years in a country district. Certain principles must be obeyed, but I can tell you that I have maintained a breeding kennel for 16 years now free of distemper apart from once when I introduced it at vaccination. There are always 30 to 50 immune bitches on the premises which go away to be mated, are kept in infected kennels, often for a period of five to six weeks, and return to whelp. They do not act as carriers. There are always about 50 puppies on the premises and these remain until they are from 8 to 12 weeks old. Visitors are not allowed to touch the puppies, stray dogs are wired out, and all bitches must be immune. The rest is simple.

It may be argued that puppies at that age have some maternal immunity, but I have found that they are easily infected if taken away and given virus by injection.

I do not think that worms, or even lice or fleas, can be incriminated as possible carriers of virus distemper. We take reasonable precautions, and whilst fleas and lice are rare in our bitches one cannot deny their presence sometimes, and worms certainly are often present in the brood bitches. It would seem impossible to have escaped infection for so many years if these parasites played any important part.

I believe, with the authors, that the rapid Carré type of the disease does not leave carriers. I also agree that the encephalitis type of case may linger on for weeks and survive with the usual protruding eyeballs and slight loss of co-ordination. I am not at all surprised that virus could be recovered from the brain of such a case after some weeks of apparent convalescence, but although I doubt if the dog could shed the virus, I would most certainly isolate him from all contact with youngsters.

One must admire the authors for their attempts to type viruses into A, B, C and in view of all that has gone before, one must hope that they will still be of the same mind a few years after this. They leave the back door open and I suspect they will use it. I, for one, disagree with them when they say that it can be concluded that progressive and continuous changes have been occurring in strains of distemper virus. As the disease affects the animals under my care, I can only say that epizootics change in character, but there is no progression towards differences in the disease. Rather is there a greater appreciation of the presence of certain symptoms. We know of the work described by Mansi, who found that all " four strains " which he tested were indistinguishable in the cross immunity tests. Similar results were obtained by Gillespie & Koprowski. This was not the experience of MacIntyre, Trevan & Montgomerie. Nevertheless, whatever the explanation, I find that any dog recovered from clinical distemper is immune to any further distemper-like illness by natural infection so long as he remains on our premises and that is up to 12 years at a maximum. This appears to me to be one of the strongest arguments in favour of condemning the theory of progressive changes. Hence I argue with the authors when they discuss recovery from the viremic form of the disease and conclude that proof of lifelong immunity following it is not sufficiently supported by statistical evidence. People in my position can do no more than state the facts. It may be that a continuing immunity depends on continued contact with the disease, but I maintain that under these conditions the immunity is unchallengeable by disease. One may speculate that if a recovered dog were isolated in a disease-free area for 10 years he might contract the disease when exposed after that, but the chances of any dog being so isolated under natural conditions must be very rare indeed.

Now many of you here to-day must be wondering how this excellent paper affects you who are in practice, how you are going to treat affected dogs, and, perhaps best of all, start or continue to protect young dogs from the disease.

Treatment

With regard to treatment I agree with the authors that antiserum has little value. Nevertheless, in the case of a very valuable dog, in contact with the disease, whose temperature is being taken regularly, it might be worth while injecting 1 c.c. per lb. bodyweight should the temperature rise. I have a record of one such case which responded to treatment without exhibiting further symptoms, but there are many others which received up to two-thirds of that amount which did not respond at all. Again, I have seen no beneficial results from the few cases which have been given live virus as treatment, but here I should say that the virus probably was not given at an early enough stage for the results to be properly assessed. Antibiotics and innumerable drugs have been tried by myself and my colleagues all without leaving the impression that any one was capable of arresting the onward course of the disease.

Immunisation

This is quite the most intriguing aspect of distemper and one which, when mastered, will give most benefit to dogs throughout the universe. I cannot go into all the history of vaccination nor all the beliefs about it. I know that dogs have been reported successfully immunised by two injections of large amounts (40 to 50 c.c.) of formolised infected spleen tissue. Two doses of 5 c.c. commercial anti-distemper vaccine have failed in my hands to produce immunity. I know that dogs have been rendered immune by means of

egg-adapted virus, which is said to be antigenic without being infective. I have also found that some dogs are apparently immune which have been given virus serum and have not reacted at the time, or it may be that the reaction was of such short duration that it escaped the temperature reading times. My main experience, however, is that I am unable to get that amount and degree of immunity which I was able to have more than 15 years ago. It may be said that I cannot, because the disease has changed. If that is so, it is the more shame to us because we are not using the virus used 15 years ago for our work—we are using the virus which is infecting our dogs naturally to-day and I long for the harnessing of this virus so that I may use the old virus serum method with the present infecting agent.

My approach to immunisation is this. As I said earlier, in my opinion, the recovered dog is immune for life, given normal canine habitable conditions. This dog has reacted physically to natural infection and my quest is for a virus which will cause a physical (temperature) reaction of 103° or more, and for accompanying antibody treatment to control that reaction so that it may be of the shortest possible duration.

I prefer virus serum to vaccine virus because there is no waiting period between doses—but only for that reason. I prefer a live virus to a non-infective one because I believe that a temperature rise following injection is the only proof that we have in practice that the virus is actually alive. In too many cases, one surmises, both here and in America, dead virus has been injected with no antigenic value at all. For that reason, too, I have no great enthusiasm for an egg-adapted virus, as one has no evidence that the material is still viable. Risk there must be if live virus is being used, but it is essential that isolation should be practised in order to minimise it. For all these dogs which have been immune after non-reaction following vaccination by various methods, I have had just as many which behaved in the same way at vaccination time and proved later to be still distemper susceptible. Many lost their lives.

In case there is any doubt in your minds I must say I have no experience of the product Epivax, and the material I used was never marketed and was purely experimental. Nevertheless, a great deal was expected of it at the time.

During two years' work the majority of dogs which I have rendered solidly immune have been those which reacted to live virus ; and but for them I should have had a very poor report to submit. Always, however, I have had a real difficulty in getting virus material certain to be capable of infecting.

In 1952 I vaccinated over 100 dogs with the experimental egg-adapted virus only. The percentage immune was 42·5 and those taking distemper 57·5 per cent. The death rate was 12·8 of the total.

In 1953, as a result of my repeated requests to Messrs. Larin & Hodgman, the latter agreed to produce for me a live ferret spleen virus which would, we hoped, give a reaction after injection, and serum to be given with it, or soon afterwards.

I have had a hard fight against the infectious hepatitis factor, not so much in the dogs as in Messrs. Larin & Hodgman. I have had to contend with the absence of reaction when it was expected that I should have it. Useful information has been exchanged between us and experiments have been carried out by Larin in the purification and even in the "typing" of virus. A factor which always disturbs us in the use of serum is the possibility of the presence of another virus as well as the antibody to distemper virus. I was therefore very pleased to make use of gamma globulin fraction of the serum and used it diluted in 1 to 2 c.c. distilled water in lieu of 10 c.c. serum.

Results were better than in the previous year. In all there were vaccinated (adults) 235, 27 of which required treatment. Three died. There were :—

Reactors	148 (63 per cent.)
Non-reactors	87 (37 per cent.)
Total taking distemper ...	42 (17·9 per cent. of total)
Reactors taking distemper ...	3
Non-reactors taking distemper	39
Total number died or destroyed	14
Total number died (all causes)	17 (7·2 per cent.)

An interesting feature of the breakdown in immunity process was that 21 of the 42 took distemper in one of our places 50 miles away, while the other 21 took it on our home premises and were nursed in our hospital. In the distant area, 14 of the 21 were from one batch which were vaccinated only a month before by Dr. Larin's purified virus and gamma globulin. They were all non-reactors. Of the distant 21 there were eight deaths, six of which were from pneumonia and exhaustion, one was destroyed following fits, and one destroyed paralysed. In no case was there any visible evidence of hardened pads.

On the home premises there were six deaths, *all* with hard pads. We therefore had the spectacle of two outbreaks, each occurring at the same time, affecting similarly vaccinated dogs, but the outbreaks differing in type, and 41 of the 42 being non-reactors. All other reactors on both premises remained immune.

A loss of 7·2 per cent. in 235 cases cannot be considered satisfactory, and I look forward very anxiously for still better results. The Animal Health Trust cannot spend too much time—so far as I am concerned—in attempting to elucidate still further the distemper problem and publishing their views on how to prevent the disease in our dogs. If they believe that infectious hepatitis is a big factor in determining life or death in distemper-affected dogs then all I ask is that they, and others interested, do work on hepatitis vaccine preparation, so that we can protect against that disease before we vaccinate against distemper.

Dr. Larin, replying to Mr. Bateman, said that with regard to various types of the virus, he maintained that this was not entirely a theoretical problem but a practical one as well. He referred to the disease in the ferret as described by Laidlaw & Dunkin. If a ferret inoculation did not result in the "classical" disease, one could not say that it was not distemper. With regard to mutation of the virus, it was not a term which he liked since a true mutation presupposed the existence of genes. The present investigation showed that A type virus was isolated only in the minority of cases.

With regard to temperature reaction, it was not to be expected that an equilibrium between antibody and virus should always be achieved. Miracles of this sort could not be expected. Where a "physical" reaction occurred it meant active disease. Mr. Bateman's approach was not the same as that which had to be adopted with a pet dog. He could afford to take a risk with greyhounds. It was probably true to say that a virus disease left a more staunch immunity than a vaccine.

Dr. Mansi wished to thank the speakers for their clear paper. The subject had been covered from a few angles. There were two points of view in studying this virus problem. From the research workers' aspect experimental animals under their disposal were inoculated with different known strains of virus and left to show precise symptoms with or without fatal results. When necessary animals were destroyed to obtain more information. On the other hand from the practitioners' point of view, they were handicapped in obtaining a definite diagnosis at an early stage of infection and it would be embarrassing if their treatment failed. Experience

had shown how clinical diagnosis only could be misleading.

In his view, the ferret was the best laboratory animal they had at their disposal. Dr. Mansi thought it even more susceptible than the dog, because mortality was so very high. The introduction of new strains from the field varied and some gave typical Laidlaw & Dunkin reactions in the ferret. The strain of Laidlaw-Dunkin they used had not varied since 1925 because a uniform method of injection was used. If the route was varied, different symptoms were seen as happened with " Glasgow S123." He would like to know why Group C differed from A and B and what happened to Group B after a few passages.

Dr. Mansi was interested in the question of vaccination. When a new vaccine was developed everything was taken into consideration in the laboratory, but once it left the laboratory, then it became exposed to different conditions. It could be damaged by heat, light or by incorrect administration. He would not agree that avianised vaccine did not give satisfactory results. He had done some work in this field and found that if the egg-vaccine was well prepared then it was satisfactory. It did not, however, behave uniformly in all eggs. During the last six years, he had not found it possible to get the virus to grow after five or six generations. Recently they had used different eggs and had obtained very good results.

Dr. Larin, in reply to Dr. Mansi, pointed out that he had said that the ferret was less susceptible to Group C virus than to Group B or A. He agreed that a strain of virus labelled " Laidlaw & Dunkin " virus had not changed since 1926, but he did not think that it was possible to draw conclusions on one strain propagated under laboratory conditions. The most important property of the canine distemper virus was that it was not homogeneous. Dr. Mansi's own strain, Glasgow S123, had changed after five or six passages. The B Group virus was very unstable and appeared to revert easily either to A or C.

With regard to immunity, he was not certain, but was of the opinion that it was probably wrong to compare the mechanism of immunity response in the ferret with that in the dog.

He had no practical experience with egg-adapted virus and would ask Mr. Bateman to answer this point.

Mr. Bateman intimated that he had been given experimental material and had lost no time in using it. He had not found it to be of any value. He went on to ask Dr. Larin whether the virus would always come true to type after passage through many ferrets.

Dr. Larin, replying, referred to the fact that one could not see the virus and must, therefore, employ biological methods. Without the ferret it was not possible to know whether the virus existed. Laboratory confirmation of the existence of canine distemper virus was possible only on ferrets. If laboratory workers stuck to the original Laidlaw & Dunkin definition their ferrets would be often discarded. This would result in a failure to establish a diagnosis, probably in 50 to 60 per cent. cases.

Dr. Mansi pointed out the importance of ferrets in the diagnosis of both canine distemper and canine virus hepatitis ; the ferret being insusceptible to the latter.

Mr. Hodgman said that he wished to emphasise the fact that the classification of A, B and C types was instituted for their own use and convenience in the laboratory, and was not meant for general use.

Dr. Montgomerie expressed his pleasure at having the opportunity of hearing this paper, for he appreciated the importance of the work on which Dr. Larin was engaged. Dealing first with the plurality of the virus, Dr. Montgomerie wished to know how Dr. Larin defined the Laidlaw-Dunkin virus. These workers had laid down very clearly the characteristics of the virus with which they worked, particularly the reaction which it produces in susceptible puppies and in ferrets. Following on the work of Laidlaw & Dunkin very many strains of virus were isolated at the Wellcome Laboratories during the period while Dalling worked there and up to the early days of the war. During the war there had been much less of this work, but in every case the virus isolated from suspected distemper cases had behaved in the characteristic fashion described by the original workers. Thus in the dog some three or four days after inoculation the characteristic diphasic temperature reaction was observed and in ferrets typical symptoms were seen about the eighth day and death occurred within 12 days. The ferret was used for the determination of virus in suspected material, generally two ferrets were inoculated with tissues from each of two or three organs, often including the brain. In this use of ferrets for diagnostic purposes the animals were observed for 15 days, survival indicating that virus was not present. To confirm that the ferrets were not immune all survivors were injected on the 15th day with ferret tissue known to contain Laidlaw-Dunkin virus. Apart from the rare occurrence of an immune animal, all these test ferrets had reacted typically to the challenge with known virus. When this work was re-started in full measure after the war there were occasions in which ferrets injected for diagnosis survived to the 15th day but thereafter showed symptoms of distemper within a few days of the inoculation of known virus. This was the first indication they had had that something unusual was happening and it could only be explained by assuming that the first inoculum contained a distemper-like virus which was atypical. Thereafter the period of observation before challenge was extended and some groups of ferrets inoculated with material from clinical cases or carcases showed no symptoms until even the 32nd day. Furthermore, the virus obtained from these cases did not give the characteristic diphasic reaction when inoculated into susceptible puppies. That seemed a good ground for saying that another virus or a variant of the Laidlaw-Dunkin virus was at work. It was at about this time that the term " hard pad " came into use in the laboratory because many of the cases from which this new type of virus was isolated had shown a syndrome different from that generally associated with distemper and, in particular, a distinct keratinisation of the foot pads. Some might well regard it as unfortunate that this piece of laboratory slang had slipped into general use both clinically and scientifically. On the other hand, if one studied American literature of some years ago, one became convinced that diseases described as distemper of the dog in some of these articles were not the condition described by Laidlaw & Dunkin but were of the " hard pad " type.

Mutation, surely, was something which one should expect, for it did occur even among the bacteria. Dr. Montgomerie was, then, very ready to accept the indications which Dr. Larin's work had unearthed and his particular concern at the moment was to know to which of Dr. Larin's groups the true Laidlaw-Dunkin type of virus should be assigned.

Immunity to virus diseases was not well understood except that it seemed essential that living virus be given a chance to invade the tissues of the animal before a solid immunity could be established. The difficulty was to control that invasion to avoid the production of disease. The use of serum after the injection of virus had been practised for many years and it had always been assumed that in this method the virus was given the opportunity to invade tissue to some extent before further invasion was cut short by the viral antibodies in the serum. On the other hand, the vaccine-virus method meant that a temporary immunity was already established by vaccination before the virus was administered

and some might be surprised that in these circumstances the virus was able to invade tissue and establish a good immunity. Mr. Bateman had always considered that if there was no real response to the injection of virus—no temperature reaction—there would be no solid immunity. Others had not agreed with this view.

The use of egg-adapted virus had been mentioned. It appeared to have the great advantage that one did not need to interfere with the injected virus by using serum to control it or having some level of immunity before it was injected. It could, then, have the opportunity to produce solid immunity. In laboratory experiments, after the initial difficulties of production had been overcome, the egg-adapted virus had shown up favourably. Now that it was coming into widespread use in the field there had been occasional reports of dogs injected with it showing serious reaction, almost always of the nervous type. In some of these cases the reaction had started within 24 hours of the injection, in others some weeks later. This might indicate that the egg-adapted virus could revert to a virulent type in the dog but so far they had no evidence that this could happen. It seemed more likely that these dogs in which reaction occurred were already carriers of a virus in the sense that it was locked up in nervous tissue, probably in the cranial cavity. Then it might well be that the injection of almost any substance could act as a trigger mechanism and set up disease. It was interesting to note that distemper virus had been recovered from the brain of dogs long after apparent recovery.

Dr. Montgomerie supported the use of the ferret as the best laboratory animal available for the investigation of distemper and immunising agents. Until egg-adapted virus was introduced there was no method of immunising the ferret. Now each batch was injected into the ferret to ensure that it had lost its virulence and that it did immunise. With properly prepared egg-adapted virus one regularly got a solid immunity in the ferret—an animal which was even more susceptible than the dog.

Dr. Larin, in reply to Dr. Montgomerie, said he was grateful to Dr. Montgomerie for the data he had given, but he wished to refer again to the table of virus types where their properties were clearly described. With regard to inoculations of ferrets with Group C virus, 50 per cent. of the animals survived, and some of the survivors appeared to be immune and some not to challenge inoculations with Group A virus.

Mr. Hodgman observed that, with regard to the virus being held in brain tissue, when examining apparently normal and susceptible dogs he had often seen an injection of the sclera. This, in his opinion, if not diagnostic suggested that these animals were in an encephalitic state. Such dogs, if inoculated against distemper had often subsequently developed nervous manifestations similar to those one saw in the syndrome called " hard pad " disease.

Regarding the typing by Dr. Larin of A, B and C viruses, on many occasions whilst investigating epidemics he sent material to Dr. Larin for diagnosis, and at the same time had made a mental note as to what type was likely to be found. In so far as A and C types were concerned, it was interesting to note that more often than not the clinical picture did suggest the type of virus which was eventually found, and although not wishing to put this forward as a diagnostic measure he thought it worth mentioning.

Brigadier A. G. Heveningham wished to thank the President and members of the Midland Counties Veterinary Association on behalf of Colonels Coulden, Holland and himself for their very kind invitation to the meeting. The excellent paper given by Dr. Larin was one of the greatest interest and importance to the Army, and would be eagerly read by Officers of the Corps both at home and overseas.

He was in no way prepared to enter into discussion on a paper of such high scientific level. He confessed to being somewhat confused and wanted to know whether it had been established that the two types of distemper virus could be present in the body of the dog at the same time, and the stronger overcome the weaker.

When it came to an outbreak, was it a case of the distemper virus launching the attack on the body defences with leptospirosis as a secondary invader ? What protective measures could be taken ?

Dr. Larin, in reply, said there was probably a relationship between the virus types described in this paper, but it was difficult to say to what degree this relationship existed. With regard to leptospirosis and distemper, if both were present at the same time nothing was of any value. Vaccines could not be expected to be of any use and he would think that the best line was serum and chemotherapy.

Major F. Chambers said he could not but feel impressed by what he had heard, but although he realised that the egg-adapted virus gave an easier method of preparing an efficient vaccine he still had faith in the now old-fashioned vaccine-virus method.

He had kept a careful record of every dog immunised or injected since the method was put on the market, and only five had died from distemper after being injected. Three of them were probably infected when the virus was put in. This was the record up to early last year when the good record was spoilt by an outbreak in a foxhound kennel. This was due to Type C of the virus and out of 35 cases seven died or were destroyed on account of fits.

It was his practice to use the virus as soon as received, and if there was any reason for delay it was put in the deep freeze. Virus left on the office mantelpiece over a hot fire for five or six days was not going to give much immunity. There was no doubt that quite a percentage of dogs were naturally immune.

Field conditions were not the same as in the laboratory or in greyhound kennels, and the practitioner could not afford to use a vaccine or a method which might give rise to an attack of distemper a few days after inoculation.

Dr. Montgomerie said he appreciated the disappointments which were being caused by the failure to meet all the demands for egg-adapted virus. The difficulties attending vaccine production could not well be appreciated by those who worked in the field. In this case it was thought that a sufficient stock of material was available when the product was introduced, but the demand for it had far exceeded the expectations and difficulties in maintaining the level of potency which had been adopted had been greater than was anticipated.

Dr. Larin pointed out that the classification of virus types he had given was a tentative one for laboratory purposes only. Group A resembled the Laidlaw & Dunkin virus, Group B was intermediate, and Group C was at the other end of the scale.

Mr. R. O. W. Walpole wished to know what were the sources of infection in clinically inapparent cases. He also wished to know whether or not there had been a change in the subject rather than a change in the virus. Breeding was now definitely along intensive lines with in-breeding not at all an uncommon thing these days. Might this not explain the increased neurotropic affinity of the virus ?

Dr. Larin, replying to Mr. Walpole, asked whether the speaker was enquiring how distemper could be spread from sub-clinical cases. The virus could be present in kidney or lung tissue and could be excreted. The only knowledge of sub-clinical cases was the evidence of solid immunity to

the virus in adult dogs which had not been seen ill with distemper. Thus, the only possible conclusion was that the specific immunity developed as the result of sub-clinical infections.

Mr. E. Weissberg : Everything they had heard that day confirmed what was already known in practice with regards to variation in the virus. Sometimes dogs responded, sometimes they did not. He wished to ask Dr. Montgomerie a practical question. Which type of virus was used in the preparation of egg-adapted virus ?

Dr. Montgomerie said it was difficult to say with accuracy what type of virus they had in the egg-adapted material, because it had been so adapted to the egg that it would not infect ferrets and its place in one of Dr. Larin's groups could not be determined. All he could say was that the egg-adapted strain had regularly immunised ferrets and dogs against the true Laidlaw-Dunkin virus and also against one which gave rise to the " hard pad " type of syndrome.

A Speaker wished to know what was the best procedure to adopt with the in-contact non-immunised dog. Should one use canine distemper serum and an egg-adapted virus vaccine ?

Dr. Larin replied that a general answer was impossible to give. Each case must be judged on its own merits by the veterinary surgeon concerned. The answer would vary in each case. If it were a pet dog he would not use the active virus, he would probably use egg-adapted virus or vaccine, or in all probability he would not immunise actively at all, and would rely on passive immunisation.

A Speaker : Which anti-serum would they recommend ?

Dr. Montgomerie said he believed the best procedure to adopt with in-contact, non-immunised dogs was to insist on routine twice daily temperature taking, warn the owner of the risk and inject " Epivax " without delay. Thereafter the temperature taking procedure would determine the need for the use of antiserum and allow its use at the most opportune time.

Mr. Bateman said he would use 10 c.c. of anti-serum followed by 20 c.c. a week later.

Dr. Mansi referred to a question which had been put earlier, in which it was said that a dog had received 10 c.c. of anti-distemper serum and recovered. Another dog which was showing high temperature and acute tonsilitis when given 10 c.c. of anti-distemper serum did not show any response and was seriously ill and then recovered. He maintained that the latter dog's case had not been distemper and was very probably a canine virus hepatitis.

In the light of this remark, with regard to the best treatment for healthy in-contact cases, he would use combined canine distemper and canine virus hepatitis immune serum followed a fortnight later by a simultaneous inoculation of avianised canine distemper virus subcutaneously and the instillation of canine hepatitis virus in the conjunctival sac of the dog. The latter combined method had proved effective under experimental conditions, for active immunisation against both virus infections.

Dr. G. B. Brook said that in 1926 chorea in the dog had always been considered to be the end of the picture. In 1944 they found themselves completely wrong. Twitching was frequently the first chapter leading to mental disintegration. They always feared a central nervous assault in practice and they thought they could get away with all types of distemper provided that the dog escaped a cerebral attack. He suggested that involvement of the central nervous system was rare in short-nosed dogs, and wondered whether immunisation via the central nervous route had been tried.

Dr. Larin, in reply to Dr. Brook, agreed that 25 years ago the nervous syndrome in distemper was observed very rarely. It now seemed that the picture was reversed, and the central nervous system involvement was often seen first. He regretted that he had no experience of Dr. Brook's suggestion with regard to immunisation via the central nervous system.

ABSTRACT

Neurological Examination of the Dog*

This is a useful article well illustrated by a series of typical cases of canine nervous disorders. The author subdivides the central nervous system into cranial nerves, cerebrum, cerebellum, spinal cord and spinal nerves. Under each of these headings is given a résumé of the function of the particular area or part and an indication of the type of lesion and its distribution which may be encountered. The effects of such lesions are then discussed. Lesions of the cerebrum may produce excitement, irritability, hysteria, lassitude, depression, coma, impaired intelligence, inco-ordination of movement and convulsive seizures. Structural changes in the cerebellum may produce disturbances of posture and of the phasic activities initiated by other parts of the central nervous system. Changes in the cranial and spinal nerves may produce effects in the areas supplied by these nerves. The effects of spinal cord lesions are discussed after the two main functions (reflex centres and conduction pathways) have been described.

The author suggests that when an animal is presented with neurologic disease three main questions should be answered. These are (1) Where is the lesion ? (2) What is the lesion ? (3) What causes it ? Information is gained by observing the animal, its gait and the position of the head and the limbs. The state of consciousness, the general body condition and the state of the skin and hair may all provide useful information. The author describes a number of reflexes which have been found to be useful in neurological examination. These reflexes may be divided into the spinal reflexes, *e.g.*, the flexor or extensor ones and the attitudinal or postural reflexes which enable the animal to maintain normal attitudes under varying conditions.

Neuropathology, from the clinical standpoint, may be divided into five main groups comprising inflammation, metabolic disorders, congenital abnormalities, trauma and neoplasms. Examples are given of each of these types and the essential symptomatology is discussed. In meningitis the symptoms are high temperature, hyperaesthesia and cervical rigidity ; in encephalitis the author considers inco-ordination, convulsions, head tilt, opisthotonus, psychic changes, blindness, deafness, pain, paraplegia and tics to be the important signs. Metabolic disturbances produce nervous symptoms, the fundamental change in all cases is hyper-excitability. Congenital abnormalities produce signs which depend upon the area of brain involved, *e.g.*, cerebellar disease will produce ataxia whereas congenital optic nerve changes may result in blindness. With trauma there may be inco-ordination, head tilt, nystagmus and prostration. In these cases of cranial trauma often there may be important changes in the temperament of the animal, frequently the damaged area is found to be in the base of the brain involving areas of the frontal lobe. The author discusses the symptomatology of cranial tumours and tumours of the spinal cord and concludes his survey by including a number of miscellaneous conditions, such as syringomelia and arthritis of the vertebral canal.

A. R. J.

* **Neurological Examination of the Dog with Some Clinico-Pathological Observations.** McGrath, J. T. (1953). *Vet. Ext. Quart. Univ. Pa.* No. 132.

B.V.A. ANNUAL CONGRESS AT TORQUAY

SEPTEMBER 19th to 25th, 1954

Provisional Programme

N.B.—All meetings will take place in Torre Abbey unless otherwise stated.

SUNDAY, SEPTEMBER 19th

8.00 p.m.
"At Home," given by the Western Counties Division, B.V.A.

MONDAY, SEPTEMBER 20th

10.00 a.m.
Civic Welcome by His Worship the Mayor of Torquay followed by opening of Congress by the Rt. Hon. the Earl Fortescue, K.G., P.C., C.B., O.B.E., M.C.

10.30 a.m.
Opening of Exhibition of Surgical Instruments, Drugs, etc., by the President, B.V.A. (Spanish Barn, Torre Abbey).

12 noon
Films.

1.00 p.m. LUNCHEON

2.30 p.m. *Main Hall*
Paper : "Meat Inspection Abroad," by Mr. Horace Thornton, B.V.Sc., M.R.C.V.S., D.V.H. (Newcastle upon Tyne).
Openers : Mr. R. H. Heywood, B.V.Sc. (Australian Meat Board);
Mr. C. V. Dayus, M.R.C.V.S. (Agricultural Liaison Officer to the High Commissioner for New Zealand);
Lieut.-Colonel Rolland O. Scott (U.S.A.F. (V.C.)).

8.30 p.m.
Civic Reception and Dance, given by the Mayor and Corporation of Torquay in the Marine Spa.

TUESDAY, SEPTEMBER 21st

9.30 a.m.
Coaches depart from Torre Abbey for Dartington Hall.

10.00 a.m. View Cattle Breeding Station
11.00 a.m. View Parsonage Farm
12 noon View Barton Farm
} In three groups.

1.00 p.m. LUNCHEON

2.30 p.m.
Paper : "Present Position with Regard to Bovine Mastitis and a Review of Recent Trials of Various Antibiotics for Bovine Mastitis," by Mr. C. D. Wilson, M.R.C.V.S. (Weybridge).
Opener : Mr. J. D. Dunn, M.R.C.V.S. (Wadebridge).

2.30 p.m.
Ladies to be shown round Dartington Hall.

2.30 p.m. *For those remaining in Torquay*
Films in Main Hall, Torre Abbey.

4.30 p.m. TEA, which ladies are invited to join.

5.30 p.m.
Coaches return to Torquay.

8.30 p.m.
Ladies Guild Dance at Palace Hotel.

WEDNESDAY, SEPTEMBER 22nd

10.00 a.m. *Main Hall*
Paper : "Myxomatosis," by Mr. J. N. Ritchie, B.Sc., M.R.C.V.S., D.V.S.M. (Tolworth), in collaboration with Mr. J. R. Hudson, B.Sc., M.R.C.V.S., and Mr. H. V. Thompson, B.Sc. (Weybridge).
Opener : Professor R. E. Glover, M.A., B.Sc., F.R.C.V.S. (Liverpool).
Break for coffee in Exhibition Hall (Spanish Barn)
Followed by *Paper :* "X-ray in Clinical Diagnosis," by Lieut.-Colonel J. Hickman, M.A., M.R.C.V.S. (Cambridge).
Opener : Lieut.-Colonel R. C. Crowhurst, O.B.E., M.R.C.V.S. (Newmarket).

11.00 a.m. *Small Hall (Adam Room)*
Paper : "The Prevention of Enterotoxaemias of Sheep," by Dr. R. F. Montgomerie, B.Sc., F.R.C.V.S., and Mr. A. Thomson, M.R.C.V.S. (Beckenham).
Opener : Mr. D. Harkness, M.R.C.V.S. (South Molton).

12.30 p.m.
Meeting of 1953–54 Council, B.V.A. (Lounge).

1.00 p.m. LUNCHEON

3.00 p.m.
Annual General Meeting (Main Hall).

7.00 for 7.30 p.m.
Annual Banquet at the Palace Hotel.

THURSDAY, SEPTEMBER 23rd

10.00 a.m. *Main Hall*
Paper : "Present Disease Position in the Poultry Industry," by Dr. R. F. Gordon, M.R.C.V.S. (Houghton, Hunts).
Opener : Dr. W. P. Blount, F.R.C.V.S. (London).

10.00 a.m. *Small Hall (Adam Room)*
Paper : "Fungoid Infections in Animals," by Dr. G. C. Ainsworth, B.Sc. (Exeter).
Opener : Mr. C. V. Watkins, M.R.C.V.S., D.V.S.M. (Starcross).
Break for coffee in Exhibition Hall (Spanish Barn)

12 noon or immediately following previous paper, *Main Hall*
Paper : "Summary of the Known Functions of the Adrenal Gland," by Mr. J. L. McGirr, B.Sc., M.R.C.V.S. (Weybridge).
Opener : Dr. W. R. Kerr, B.Sc., M.R.C.V.S., D.V.S.M. (Belfast).

1.00 p.m. LUNCHEON

2.30 p.m.
Films, Golf, Tennis, Bowls, etc.

8.00 p.m.
Theatre bookings have been made.

FRIDAY, SEPTEMBER 24th

10.00 a.m. *Main Hall*
Short Papers : "Pig Rearing," by Mr. J. W. Reid, N.D.A. (Starcross); "Pig Nutrition," by Dr. D. E. Tribe, B.Sc. (Bristol); "Pig Diseases," by Mr. H. F. Hebeler (Langport).
Opener : Professor F. Blakemore, D.V.Sc., B.V.Sc., M.R.C.V.S., D.V.S.M. (VICT.) (Bristol).
Break for coffee in Exhibition Hall (Spanish Barn)

11.00 a.m. *Small Hall (Adam Room)*
Paper : "Neoplasia in the Dog," by Mr. E. Cotchin, M.R.C.V.S. (London).
Opener : Mr. J. Allcock, B.V.Sc., M.R.C.V.S. (Bristol).

1.00 p.m. LUNCHEON

2.30 p.m. *Main Hall*
Paper : "Scientific Trends in Milk Production in Great Britain," by Dr. Joseph Edwards (Milk Marketing Board).
Opener : Mr. John R. Currie, M.S., B.Sc., N.D.A., N.D.D. (Dartington Hall).

2.30 p.m. *Small Hall (Adam Room)*
Paper : "Hereditary Conditions in the Dog," by Dr. G. B. Young, M.R.C.V.S. (Edinburgh).
Opener : Miss J. O. Joshua, F.R.C.V.S. (Finchley).

8.30 p.m.
President's Reception and Dance at the Palace Hotel.

SATURDAY, SEPTEMBER 25th

10.00 a.m. *Main Hall*
Closing Meeting of Congress, followed by First Meeting of Council, 1954–55.

CLINICAL COMMUNICATION

SOME OBSERVATIONS ON THE RETENTION OF THE FOETAL MEMBRANES IN DAIRY CATTLE AFTER THEIR FIRST PREGNANCY

C. R. COID AND A. McDIARMID

AGRICULTURAL RESEARCH COUNCIL, FIELD STATION, COMPTON, BERKS

INTRODUCTION

Reference has been made by several authors to the incidence of retention of foetal membranes in the bovine. The following incidence figures are quoted by Benesch & Wright (1950): 11·7 per cent. (Palmer, 1932), 4 to 5 per cent. (Götze, 1941, 1942) and 8·3 per cent. (Kennedy, 1947).

Methods of treating cases of retention have been widely discussed and some veterinary surgeons are of the opinion that manual removal of the foetal membranes is often ill-advised. This view was expressed by Gould (1944) and also by Lang (1944).

Benesch & Wright quote reports of successful treatment by methods other than manual removal.

ANIMALS UNDER OBSERVATION

The cattle on which the following observations were made were assembled for a long-term experiment on brucellosis. They comprised a representative sample of cattle from Ayrshire and, apart from a short period at grass after weaning, they were housed, in units of 12, in an isolation compound.

All were tuberculin tested and the majority vaccinated with *Br. abortus* S.19 vaccine. None of these animals was infected with a virulent strain of *Br. abortus* during the course of observation.

At parturition, in addition to routine agglutination tests for brucellosis, material was examined microscopically for the presence of *Br. abortus*, *V. foetus* and *Trichomonas foetus*. No evidence of these infections was detected.

Incidence of Retained Foetal Membranes

Animals were considered to have retained foetal membranes if complete expulsion did not occur within 72 hours after parturition.

The total number of cattle observed was 382, and of these 14 (3·7 per cent.) retained the foetal membranes. One of the latter, No. 1, aborted at seven months; this proved to be of a non-specific nature.

Treatment Adopted

In the one case of abortion, the foetal membranes were removed manually after seven days' retention. This animal is not considered in the remainder of this communication. Of the 13 animals which were not subjected to any manual interference, eight were treated with 10 mg. stilboestrol dipropionate intramuscularly and two were given a further injection after an interval of three days. The remaining five animals did not receive any treatment. Details of the treatment and condition of each animal at the time of retention are shown in the table.

Milk Production

Milk recording commenced the fourth day after parturition and the milk yield per cow during the following 14 days is shown in the table.

In the affected cattle, the average milk yield (342 lb. ± 55) was slightly less than that of 50 others (364 lb. ± 89) in the same herd, but which did not retain the foetal membranes.

The comparison shows that there is no significant difference between these two samples ($p > 0{\cdot}4$).

Subsequent Breeding History

After the first parturition, the cattle were not served before a pre-determined date in order to conform with the original plan of the brucellosis experiment. The average number of services required for the subsequent parturition was 1·2.

DISCUSSION

According to Benesch & Wright, it is generally believed that retention of the foetal membranes for longer than 12 hours in the bovine should be considered pathological. If every case after 12 hours' and under 72 hours' retention had been recorded in the cattle observed, then the incidence of retention would have been higher than 3·7 per cent., though no exact figures can be quoted.

During the time of retention, no animal was seriously ill, though some, as shown in the table, lost their appetite and consequently showed a slight loss in condition. The milk yield during the period of retention did not seem to be seriously affected, and the average number of services per calf produced at the next parturition was satisfactory.

As a result of the above observations it was concluded that manual removal of the foetal membranes, though perhaps desirable for aesthetic reasons, is not essential.

RETENTION OF THE FOETAL MEMBRANES IN DAIRY CATTLE AFTER THEIR FIRST PREGNANCY

Number of animals	Number of days foetal membranes retained	General condition during retention	Treatment	Milk yield during first 14 days recording (lbs.)	Time between parturition and first service (days)	Number of services required for second parturition
1	7	Normal	Membranes removed manually	236	137	2
2	Over 6	Loss of appetite	None	384	69	1
3	10	Normal	"	276	75	1
4	5	"	"	308	73	1
5	5	"	"	331	79	1
6	6	"	"	415	78	1
7*	Over 6	"	10 mg. stilboestrol	291	116	1
8	" 7	Loss of appetite	"	360	220	1
9	" 6	Normal	"	276	60	1
10	" 6	Loss of appetite	"	287	102	1
11	9	"	"	390	81	1
12	Over 8	"	"	Not available	73	2
13*	9	"	"	349	84	2
14	Over 5	Normal	"	437	75	2

* Animals given a second injection of 10 mg. stilboestrol three days after first injection.

Summary

1. The incidence of foetal membranes retained for longer than 72 hours in 382 dairy cattle maintained in an isolation compound was 14 (3·7 per cent.), after their first pregnancy.

2. In one case of retention the foetal membranes were removed manually ; the remaining 13 cases were not subjected to manual interference but eight of these were treated with stilboestrol.

3. The loss of milk due to the retention of foetal membranes, in the animals observed, was slight.

4. It was found that the subsequent breeding history of the animals was apparently unaffected although the retained membranes were not removed manually.

Acknowledgments.—We wish to thank Dr. W. S. Gordon, C.B.E., the director of this Field Station, for the interest taken in this communication, and Mr. J. Elliot, manager of the isolation compound, for details of the milk yields.

References

Benesch, F., & Wright, J. G. (1950). "Veterinary Obstetrics." (London.)

Götze, F. (1941). *Dtsch. tierärztl. Wschr.* **49.** 589.

———. (1942). *Ibid.* **50.** 4.

Gould, G. N. (1944). *Vet. Rec.* **56.** 44.

Kennedy, A. J. (1947). *Ibid.* **59.** 519.

Lang, W. W. (1944). *Ibid.* **56.** 372.

Palmer, C. C. (1932). *J. Amer. vet. med. Ass.* **80.** 59.

NOTES AND NEWS

Diary of Events

June 16th.—Meeting of the North Wales Division, B.V.A., at the Glanaber Hotel, Bettws-y-Coed, 2 p.m.

June 16th.—Meeting of the Western Counties Division, B.V.A., at Torre Abbey, Torquay, 2.30 p.m.

June 17th.—Annual General Meeting of the North of Scotland Division, B.V.A., in the Northern Hotel, Aberdeen, 2.30 p.m.

June 17th.—Annual General Meeting of the South-East Midlands Division, B.V.A., at Franklins' Gardens Hotel, Northampton, 7 p.m.

June 17th.—Summer Meeting of the Herts and Beds Division, B.V.A., at Luton Hoo, 2.30 p.m.

June 18th.—General Meeting of the North of Ireland Division, B.V.A., at the Veterinary Research Division, Stormont, Belfast, 7 p.m.

June 19th.—Meeting of the Biochemical Society in the Department of Biochemistry, University of Cambridge, 11 a.m.

June 23rd.—Summer Meeting of the Royal Society of Medicine, Section of Comparative Medicine, at the Rothamsted Experimental Station, Harpenden, Herts, 10.45 a.m.

June 26th.—Joint Meeting of the Scottish Branch and the Scottish Metropolitan Division, B.V.A., in the Royal (Dick) School of Veterinary Studies, Edinburgh, 2.30 p.m.

Sept. 19th—25th.—72nd Annual General Meeting and Congress of the British Veterinary Association at Torquay.

July Meetings of Council and Committees, B.V.A.

Wednesday, July 21st, 1954:—

11.0 a.m. Organising Committee.
2.0 p.m. Home Appointments Committee.
3.30 p.m. Parliamentary and Public Relations Committee.
At 7, Mansfield Street, Portland Place, W.1.

Thursday, July 22nd, 1954:—

10.30 a.m. Veterinary State Medicine Committee.
1.45 p.m. Finance Sub-committee.
2.30 p.m. General Purposes and Finance Committee.
At 7, Mansfield Street, Portland Place, W.1.

Friday, July 23rd, 1954:—

10.30 a.m. Council Meeting.
At the Connaught Rooms, Great Queen Street, W.1.

R.C.V.S. Examinations

Monday, June 14th.—D.V.S.M. Orals and Practicals commence.
Thursday, June 24th.—M.R.C.V.S. Written Examinations.
Friday, June 25th.—Ditto.
Monday, June 28th.—M.R.C.V.S. Practical and Orals commence (Panels " A " and " B ").

* * * * *

ELECTION OF NEW COUNCIL MEMBERS, R.C.V.S.

At the Annual General Meeting of the Royal College of Veterinary Surgeons held at 9-10, Red Lion Square on June 1st, Mr. Henry Sumner was elected Junior Vice-President and the following members of the Profession were elected to the Council. The number of votes cast for each candidate are given as follow:—

Mr. A. J. Wright	**903**
Professor C. Formston	**739**
Mr. J. F. D. Tutt	**664**
Miss Joan Joshua	**663**
Mr. H. Holroyd	**663**

R.C.V.S. PRIZES

Fitzwygram, Williams, Walley and Lawson Prizes
1954 Graduates

The Prize Board for the above prizes gives notice that the subject selected for the competition open to candidates who obtain a registrable qualification in the calendar year 1954 is:—

" A discussion of the differential diagnosis of swine fever and measures for the control of the disease."

Intending candidates are referred to the regulations for the prizes on page 78 of the Register of Veterinary Surgeons (1953) and may obtain any further information from the Registrar, R.C.V.S., 9-10, Red Lion Square, London, W.C.1.

* * * * *

R.C.V.S. OBITUARY

Sheffield, Joseph (Major R.A.V.C., retd.), of Howden, East Yorks. Graduated Liverpool, December 19th, 1905. Died June 1st, 1954; aged 70 years. Cremated at Stonefall, Harrogate, June 4th, 1954.

Procter, John William (Captain late R.A.V.C.), 3, The Crescent, York. Graduated Liverpool, December 16th, 1912. Died June 6th, 1954.

FORTHCOMING CONGRESS

THE FEEDING OF FARM LIVESTOCK FOR HEALTH AND PRODUCTION

The B.V.A. is making plans for an Autumn Conference to discuss the feeding of farm livestock for health and production.

It will be held on two consecutive days—Tuesday and Wednesday, November 16th and 17th, 1954—in the South Hall, Victoria Halls, Bloomsbury Square, London, W.C.1.

The Conference will be open to members of the Association and to members of ancillary associations and trade associations. Admission will be by ticket. Arrangements for issue of the tickets will be announced. A full programme will be published in due course.

* * * * *

APPOINTMENT IN THE NATIONAL AGRICULTURAL ADVISORY SERVICE

Mr. W. P. Dodgson, Deputy Chief Livestock Husbandry Officer, has been appointed Chief Livestock Husbandry Officer in the National Agricultural Advisory Service in succession to Mr. H. L. Shrimpton, M.C., who is retiring from the public service on June 30th, 1954.

* * * * *

DRUM HORSE FOR HOUSEHOLD CAVALRY

"Bonaparte," the Life Guards' drum horse, died a short time ago owing to an attack of pneumonia with cardiac complications.

The Army Veterinary and Remount Service is now faced with the task of providing a suitable replacement, and it is anticipated that the right horse will be far from easy to find. He would have to be a clean-legged, nicely marked piebald or skewbald gelding of the heavy weight hunter type, not more than seven years old, at least 17 hands when fully grown, with a good front, and a particularly quiet temperament. The ideal markings are four white legs with plenty of white in the tail and on the upper parts of the head, neck and body.

If any member of the profession knows where there is a horse approaching these standards the Director, Army Veterinary and Remount Services, would be extremely grateful if the member would write to him at the War Office (V. & R.), Droitwich.

* * * * *

COMMITTEE TO INSPECT AGRICULTURAL COLLEGES

The Minister of Agriculture has appointed a Committee to inspect the grant-aided Agricultural Colleges. It has the following terms of reference:—

"To examine and report to the Minister of Agriculture and Fisheries on the work of the grant-aided Agricultural Colleges and to make recommendations as to the grants to be made to them for the five academic years beginning October 1st, 1954."

The members of the Committee are: Dr. K. A. H. Murray, B.SC., M.A., B.LITT., Chairman of the University Grants Committee; the Hon. Mrs. Ralph Assheton, F.L.A.S., land agent and farmer; Professor H. G. Sanders, M.A., PH.D., Professor of Agriculture at the University of Reading, Mr. G. A. Nott, farmer.

The appointment of this Committee follows the practice adopted before the war.

ADDRESSES OF DISEASE-INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

ANTHRAX:

Yorks.—The Croft, Hardraw, Hawes (May 28).

FOWL PEST:

Lancs.—Field at Warner Bridge Farm, Longton, Preston (May 29); Rhodes Farm, Wiggins Lane, Holmeswood; Russlea, Wiggins Lane, Holmeswood, Ormskirk (May 30).

Norfolk.—Park Farm, Witton, Norwich (May 31).

SWINE FEVER:

Berks.—Easthampstead Park Piggeries, Bracknell (June 1).

Cambs.—Orwell Pit Farm, Ely (June 3); R.A.F. Station, Waterbeach (June 4).

Ches.—Overton Hall Farm, Overton, Malpas (May 29); Station Piggeries, Station Road, Styal, Wilmslow; Edge Grange, Edge, Malpas (June 2).

Denbighs.—Cae Haidd Farm, Bwlchgwyn, Wrexham (June 2).

Essex.—Whelps Farm, Dunton Road, Laindon (May 29).

Glamorgan.—Tycelyn Farm, Roath Park, Roath, Cardiff; Dyhewydd Farm, Llantwit Fardre, Pontypridd (May 31).

Glouc.s.—Clingre Farm, Dursley (May 31); Newark Farm, Hempstead (June 3).

Herts.—Sewerage Field Farm, Birch Green, Hertingfordbury (June 2).

Kent.—Hill Top Farm, Lenham, Maidstone (June 2).

Lincs.—5, Victoria Road, Barnetby (June 4).

Montgomery.—Henfaes, Welshpool (May 31).

Norfolk.—Lamberts Farm, Marsham, Norwich (May 29); Old Hall Farm, Stiffkey, Wells (May 31).

Notts.—Premises off Radcliffe Road, Gamston (June 4).

Somerset.—Hayward Farm, Terry Hill Cross Roads, Radstock, Bath (May 31); Seymour Arms, Witham Friary, Frome (June 1).

Suffolk.—Kings Farm, Tannington, Woodbridge (June 2).

Surrey.—Meesons Farm, New House Lane, Salfords, Redhill; Howicks Farm, South Nutfield, Redhill (May 29); Hook Farm, Leigh, Reigate (June 1).

Sussex.—Upper Honeys Green Farm, Framfield, Uckfield (May 31); Manor Farm, Lodsworth, Petworth; Upper Wyckham Farm, Steyning (June 1).

Yorks.—Fenton Grange Farm, Church Fenton, Nr. Tadcaster (May 31); Nutter Lane Piggeries, Gomersal, Leeds; St. Margarets Piggeries, Knowles Lane, Bradford (June 1).

DISEASES OF ANIMALS ACT, 1950—GREAT BRITAIN

Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period	Anthrax	Foot-and-mouth	Fowl Pest	Sheep Scab	Swine Fever
1st to 15th May, 1954	20	—	30	—	68
Corresponding period in—1953	28	—	37	—	251
1952	44	86	6	—	57
1951	26	1	6	—	36
1st January to 15th May, 1954 ...	126	12	499	—	552
Corresponding period in—1953	378	25	247	—	1,269
1952	218	201	106	1	455
1951	220	18	463	4	316